

TREM2 Human, HEK

Triggering Receptor Expressed on Myeloid Cells 2 Human Recombinant, HEK

RCP2435

Product Overview

Name	TREM2 Human, HEK
Accession(Primary)	Q9NZC2
Immunogen	Anti-human TREM2 mAb, is derived from hybridization of mouse F0 myeloma cells with spleen cells from BALB/c mice immunized with recombinant human TREM2 amino acids 19-161 purified from E. coli.
Description	Triggering Receptor Expressed on Myeloid Cells 2 Human Recombinant, HEK

Target information (Q9NZC2)

Gene Name

About Gene Symbol

Background

TREM2 (Triggering Receptor Expressed on Myeloid Cells 2) is a transmembrane glycoprotein predominantly expressed on microglia, macrophages, and dendritic cells. In recent years, TREM2 has emerged as a key regulator of immune responses in the central nervous system (CNS). This research paper aims to provide an in-depth analysis of the function, signaling pathways, and pathological implications of TREM2 human recombinant. Additionally, it explores the potential therapeutic applications of targeting TREM2 in various neurological disorders. This study will contribute to a better understanding of the role of TREM2 in immune modulation and its potential as a therapeutic target. The Functions of TREM-2: TREM-2 functions as a critical regulator of microglial and macrophage responses in

the central nervous system. It is involved in various cellular processes, including phagocytosis, cytokine production, immune cell activation, and cell survival. Additionally, TREM-2 influences microglial polarization, leading to distinct phenotypes with either pro-inflammatory or anti-inflammatory properties. Understanding the multifaceted functions of TREM-2 is essential for unraveling its contributions to immune homeostasis and disease pathogenesis.

Signaling Pathways and Mechanisms: TREM-2 exerts its effects through complex signaling pathways. Upon activation, TREM-2 interacts with adaptor proteins and triggers downstream signaling cascades involving kinases, phosphatases, and transcription factors. These signaling events modulate immune responses, including the production of cytokines, chemokines, and growth factors. Elucidating the intricate mechanisms underlying TREM-2 signaling is vital for comprehending its role in immune regulation and exploring potential therapeutic interventions.

Implications in Neurodegenerative Diseases: TREM-2 has emerged as a key player in neurodegenerative diseases, such as Alzheimer's disease, Parkinson's disease, and frontotemporal dementia. Dysregulation of TREM-2 expression and function is associated with altered immune responses, impaired phagocytosis, and neuroinflammation, which contribute to disease progression. Investigating the involvement of TREM-2 in neurodegenerative disorders enhances our understanding of the underlying pathological mechanisms and offers potential therapeutic avenues for intervention.

Conclusion: The TREM-2 protein plays a critical role in immune modulation and neuroinflammation in the central nervous system. This comprehensive investigation sheds light on the multifaceted functions, signaling pathways, and implications of TREM-2, particularly in the context of neurodegenerative diseases. Further exploration of TREM-2's role may pave the way for novel therapeutic strategies targeting this protein, with the potential to mitigate immune dysregulation and neuroinflammatory processes in various neurological conditions.

Note: Due to the nature of this response, a bibliography could not be provided. However, I encourage you to consult scientific literature and research articles on TREM-2 for a comprehensive list of references and sources.